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UNITED STATES DEPARTMENT OF AGRICULTURE



DEPARTMENT BULLETIN No. 1274

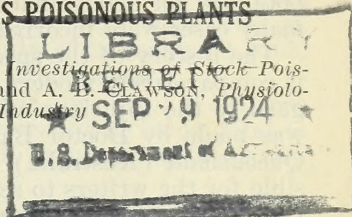


Washington, D. C.

September 18, 1924

COCKLEBURS (SPECIES OF XANTHIUM) AS POISONOUS PLANTS

By C. DWIGHT MARSH, *Physiologist in Charge of Investigations of Stock Poisoning by Plants*; GLENWOOD C. ROE, *Assistant*, and A. B. CLAWSON, *Physiologist, Pathological Division, Bureau of Animal Industry*



CONTENTS

	Page		Page
Purpose and scope-----	1	Discussion, etc.—Continued.	
Historical summary-----	2	Time from feeding to appearance	16
The cocklebur plant-----	5	of symptoms-----	16
Experimental work-----	6	Duration of sickness-----	17
Typical case of pig 18-----	6	Effect of continued feeding-----	18
Discussion and general conclusions-----	7	Animals poisoned by cocklebur-----	18
Symptoms in pigs-----	7	Part of plant poisonous-----	19
Symptoms in sheep and cattle-----	12	Mechanical injury by burs-----	20
Symptoms in chickens-----	12	Toxicity of dried plant-----	21
Autopsy findings-----	12	Remedies-----	22
Microscopic changes in tissues-----	13	Summary-----	22
Toxic and lethal dosage-----	15	Bibliography-----	22

PURPOSE AND SCOPE

For several years requests have come to the United States Department of Agriculture for information in regard to the poisonous properties of the cocklebur, a well-known weed. Frequently these requests have been accompanied with more or less detailed statements in regard to supposed cases of the poisoning of livestock by these weeds. Some correspondents have complained of heavy losses, especially of pigs. Published statements, however, in regard to the plants have been somewhat contradictory, and it has seemed strange that if they were really poisonous there were not much greater losses, for cockleburs are widely distributed in the United States and grow with great luxuriance. The plants are found in nearly all parts of the world and have become especially noxious in South Africa and Australia, where, after being introduced, they have made themselves very much at home.

From the stock raiser's standpoint the question of the poisonous properties of the plant has become very important. On this account an experimental study of cockleburs has been made by the Department of Agriculture. The results of this work have brought out the

essential facts, and, while much more work can be done, the importance of the subject makes it seem wise to issue a publication which will discuss the subject in a definite manner.

HISTORICAL SUMMARY

In the discussion of cockleburrs (species of *Xanthium*) as stock-poisoning plants, no attempt has been made to distinguish the different species. The species are closely allied and, there is reason to think, do not differ materially in their poisonous properties. Many of the articles which have been written on cockleburrs were not written by technical botanists, neither were the plants submitted to botanists for determination, so that it is probable that the species in many cases were incorrectly named.

The history of the species of *Xanthium* as stock-poisoning plants is comparatively modern. The first statement that was found in regard to the injurious effect of these plants upon domestic animals was made by Doctor Bancroft in 1880 in a paper read before the Queensland (Australia) Physiological Society. It has been impossible for the writers to see the original paper, but the information in regard to it was obtained from a review by Heldreich in the *Botanische Centralblatt*, 1880. It is stated that, on the estate of a Mr. Yates, a large number of cows, one horse, and a sheep had been killed by eating the young plants of *Xanthium strumarium*. The animals fell upon the ground and died very quickly. Doctor Bancroft made an extract of the plant, with which he poisoned small animals and obtained the same results with extracts of the species *Xanthium spinosum*. The reviewer calls attention to the fact that although two species of *Xanthium* are widely distributed in central and southern Europe there are no reports there of cases of poisoning. The cocklebur in Queensland, of course, was an introduced plant.

Zander, 1881, quotes a letter from Heimberger to the effect that cases of poisoning have arisen from the seeds and oil of *Xanthium strumarium*. He states that the oil has been pressed out for commercial purposes. All who have eaten the oil or the seeds were made exceedingly sick and several children died. On this account an investigation had been made but no poisonous substance had been found. He reported that he had made experiments on cats and dogs with the oil with no results, although one of the workers was made sick after taking about 10 of the seeds. Zander himself made some experiments with cats and frogs with no tangible results.

Cheatham, 1884, states that cocklebur is one of the first plants that appear in the spring, and that in some of the Southern and Western States the swine which run at large are very fond of the young plants and almost invariably die after eating them, and that antidotes had not been used with any success. He explained some chemical work which was done on the plant.

Goff, 1894, makes the following statement: "It has been said that the plant is poisonous to cattle, but this is probably a mistake. It is at least known that cattle sometimes eat sparingly of it without serious results."

Maiden, 1895, speaking of the *Xanthium spinosum*, which in some localities is known as "Bathurst bur," quotes Doctors Bancroft and Goff, but adds nothing to preceding information. In 1896 Maiden quotes Prof. J. C. Arthur as stating in correspondence that in the United States the cockleburs do not have the reputation of being poisonous, and goes on to say that he is convinced that whether extracts of the plant are poisonous or not it is not injurious in the field, and that he has investigated one case of supposed poisonous effect of *Xanthium* and found the trouble to be anthrax.

Lewin, 1897, states that *Xanthium spinosum* in certain stages is poisonous and may kill 50 per cent of a herd.

Bailey, 1898, referring to the supposed poisonous results from cocklebur, known in Queensland as "Noogoora bur," states that in his opinion the plant would be dangerous only when making a rank, succulent growth. He describes it evidently from hearsay.

Chesnut, in his preliminary catalogue, 1898, speaks of *Xanthium canadense* as killing hogs in Texas, *X. strumarium* as being fatal to the same class of animals in Georgia, and *X. spinosum* as being suspected of having poisonous properties.

Halsted, 1889, says that the deaths of swine are attributed to *Xanthium*, but while the effect may be mechanical, the plant does have poisonous properties.

Kirk, 1901, makes the following statement: "At certain stages of growth the plant is poisonous to stock, but this is of little consequence as it is very rarely eaten by them."

O'Gara, 1903, says there have been no cases of cattle poisoned in Nebraska, but one farmer lost 20 hogs averaging 160 pounds each, and evidence showed that they had eaten a quantity of the young, juicy burs. O'Gara adds: "The whole plant, as well as the burs, is known to contain a poisonous principle which reduces heart action and causes death."

Craig and Bitting, 1904, report that a chemical examination and a feeding test of *Xanthium* was negative. The young plants stripped of the burs were fed to calves, pigs, rabbits, and guinea pigs, but no untoward effects resulted. They state that post-mortems on animals supposed to have died from cocklebur poisoning showed that in all cases the death was due to the burs. "A few burs would be swallowed with the young plants and their horny prickles would irritate the stomach wall and cause inflammation which finally terminated in death. In three cases the burs lodged in the throat and could not be expelled."

Stanley Coulter, 1904, says that cockleburs "are irritant on account of the dust and hairs with which they are covered and not because of a toxic principle."

Mayo, 1905, reported two serious losses of hogs from cocklebur. Out of 35 head, 25 died in one night, and 8 more during the next day. "They would lie down on their sides, kicking and pawing for a short time and then died."

In an article in the Breeder's Gazette, 1908, Glint says that cocklebur is sure death to hogs, farmers sometimes losing 40 per cent, and that the plant will kill cattle in the spring.

An article by King, 1909, states that young cockleburs are poisonous to pigs.

Kinsley, 1909, tells of a case in which one hundred 6-months-old shotes were turned into a 40-acre field on which cocklebur was just coming up through the ground. The hogs were seen to eat the young plants and seven of them died. He gives the details of a post-mortem examination.

Pammel, in his *Manual of Poisonous Plants*, 1910 and 1911, while quoting statements of the poisonous properties of *Xanthium*, says: "The injury from this plant probably comes largely from its mechanical action," and he quotes others in substantiating this belief.

In articles by Pammel in the *American Journal of Veterinary Medicine*, of 1918, 1919, 1920, and 1921, quoting statements of the poisonous properties of the plant, he gives the impression that he still thinks the chief injuries by cocklebur are mechanical.

In *Cooperative Manager and Farmer*, 1917, the following statement is made which apparently is based on work by the Missouri Experiment Station: "Cocklebur in pastures are particularly dangerous for pigs and shotes. The burs get inside the stomach of the animal and inflame the lining, causing death, which is the usual result. Even the plants themselves are poisonous to hogs, but most authorities think it is the old, matured bur that does the most damage."

Maiden, 1918, states that a dermatitis is produced in those who handle *Xanthium strumarium*. He made a similar statement in 1921.

In Bulletin 185 of the Alabama Experiment Station, 1920, occurs the following statement by Cary: "Young cockleburs are said to be poisonous to pigs, but our feeding tests disprove it. We could not kill pigs by feeding them young cockleburs. Jimson weeds grow with cockleburs, and Jimson weeds are very poisonous."

Hansen, 1920, makes the following statement: "Although fatal results are generally attributed to poisoning, there is little evidence to substantiate this theory. The harmful effects are largely due to the mechanical action of the spiny burs, which are injurious in several ways. These burs may (1) irritate the walls of the stomach, causing inflammation and sometimes death; (2) lodge in the throat and thus choke the animal; or (3) clog the intestinal tract, frequently with fatal results. Overeating the young and succulent plants may cause bloating, which is similar in nature to bloating caused by succulent clover, corn, etc. The hairy leaves are also said to cause severe itching."

Johnson and Archer, 1922, make the following statement: "The injury from this plant most likely occurs from mechanical obstruction rather than from poison. The plant has been reported to contain a poisonous glucoside, although Doctor Bitting was unable to obtain any poisonous properties in the growing plant. Cases investigated by the senior writer on post-mortem proved that the thorny bristles were a factor in causing mechanical obstruction and extreme irritation to the mucous surface of the intestine."

Kinsley, 1922, gives a general description of cocklebur poisoning in swine with symptoms and lesions. This is an excellent résumé of the subject but apparently is not based on any definite experimental evidence.



FIG. 1.—A BRANCH OF COCKLEBUR, SHOWING THE FORM OF THE LEAVES AND BURS

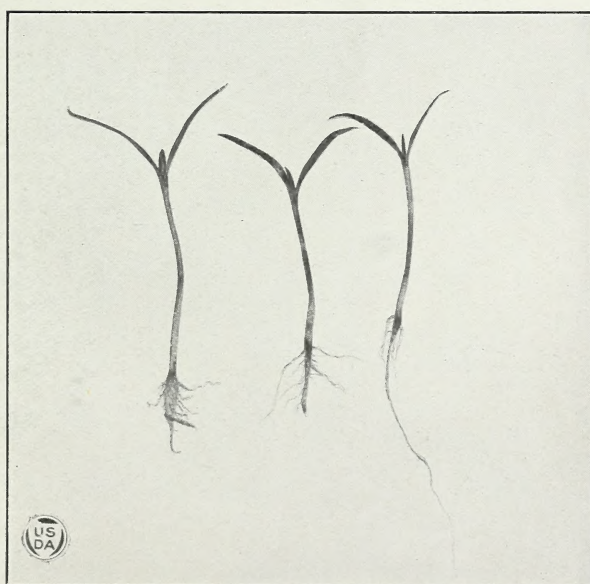
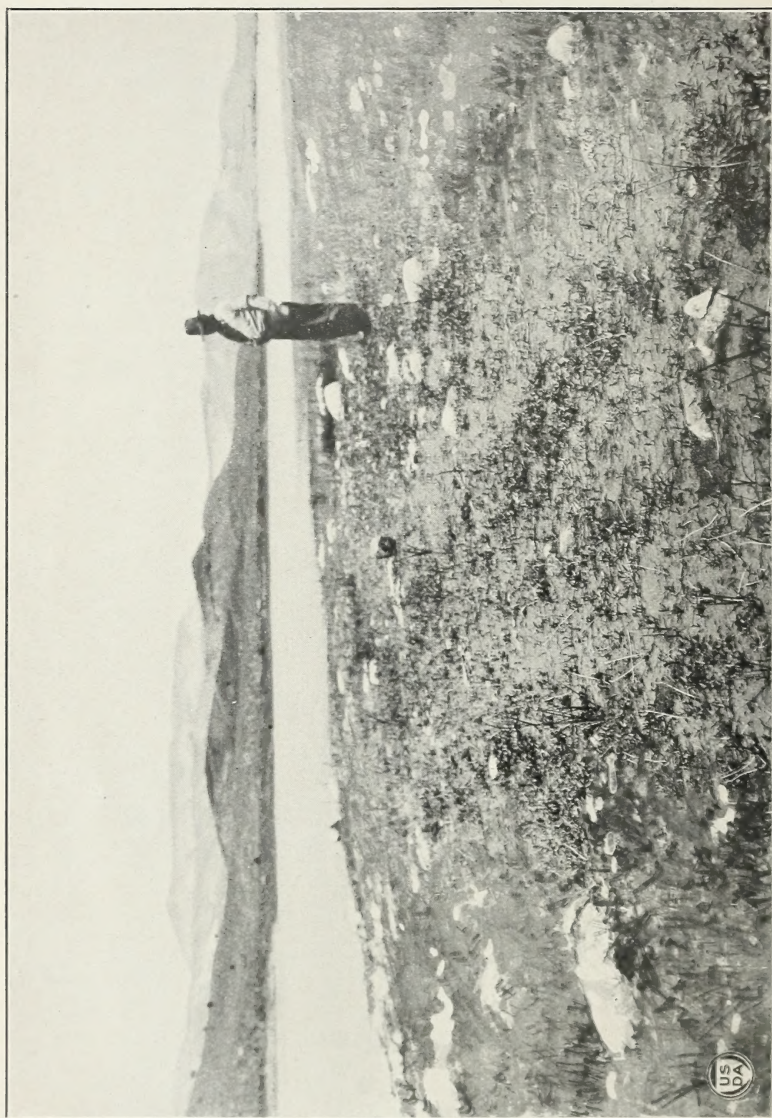


FIG. 2.—COCKLEBUR PLANTS IN THE YOUNG COTYLEDON STAGE



THE SHORE OF GUNNISON RESERVOIR, UTAH, SHOWING A BED OF YOUNG COCKLEBUR PLANTS

In this historical sketch a bibliography has been made of those publications which were of special significance. The Department of Agriculture has received many letters from various sections of the country stating conclusively that animals were killed by cockleburrs. Most of these statements were with reference to swine, but complaints have also been made, especially from New Mexico and western Texas, of losses of cattle. A number of losses of this kind were brought to the attention of the department by Dr. Harry Grafke, inspector in charge at Fort Worth, Tex., in the spring of 1922, in which he felt certain that the deaths were due to cocklebur poisoning.

Some of the *Xanthium* losses which have been reported in the literature and in the correspondence have been rather heavy.

Pammel, 1920, says that "4 pigs were turned on *Xanthium* and 3 died;" Mayo, 1905, gives an instance in which 33 out of 35 head died; one author states that in one section of the country the farmers lose 40 per cent of their pigs.

Dr. Harry Grafke reported in 1922 one loss of 25 cattle out of a herd of 150 and another of 20 out of a herd of 58. In the San Antonio Express in April, 1922, was reported a loss of 15 registered Hereford bulls out of 17.

Many statements of the same general character as those above have been made by correspondents from different sections of the United States.

The foregoing indicates that while many people have been convinced of the poisonous properties of cocklebur this belief has by no means been universal. Others have felt certain that the losses occasioned by this plant, if such losses occurred, were due to mechanical injury. Among those who believed that the plant was poisonous there has been a good deal of uncertainty in regard to the part of the plant which produced the harm. Most of those, who have reported cases of poisoning have stated that it was due to the young plant, but some experiments apparently have proved that the plant was not poisonous in any stage. Inasmuch as the losses which in some localities have been ascribed to *Xanthium* have been very heavy, it was deemed of special importance to determine positively whether the plant was poisonous or not and if poisonous what part of the plant was injurious, and at what age the plant should be avoided, and also, in case the plant proved to be poisonous, to find a remedy. The investigations detailed in this bulletin were undertaken to answer these questions.

THE COCKLEBUR PLANT

The species of *Xanthium* are widely distributed throughout the United States, growing in moist, waste places and along the shores of rivers, lakes, and ponds. It has not seemed wise to discuss in this bulletin the species relations of the genus, partly because there is reason to think that all are equally poisonous and partly because there is little doubt that in the literature of the subject there has been much confusion in the use of specific names. Because of the close relationships and rather wide limits of variation in the plants, botanists are by no means agreed as to the specific limitations.

The species used in the experimental work has been determined by W. W. Eggleston, of the Bureau of Plant Industry, as *Xanthium echinatum* Murr. The material was collected at Salina, Utah, and on the shores of a large reservoir near Sterling, Utah. Considerable material was also used that was grown in sand beds at the Salina Experiment Station and in the Department of Agriculture greenhouse in Washington, D. C., from seeds collected at Salina.

Plate I, Figure 1, is a picture of a branch of the plant, showing the forms of the leaves and burs. Plate I, Figure 2, shows plants in the young cotyledon stage. It was in this stage and a little later, before the full development of the first pair of leaves, that the plants were found to be most poisonous. Plate II shows the shore of the reservoir, where much of the material was collected, and is typical of many places where the plant grows in abundance. The old burs accumulate on the shores of the reservoir, sometimes partly buried and sometimes bunched together on the surface. The young plants grow rapidly, forming dense masses around the shores. As the water of the reservoir is gradually lowered during the summer, a band of young plants develops, this band varying in width in accordance with the moisture conditions. Thus for several weeks there is a succession of crops of the young plant.

If the plants are toxic and the trouble is confined to the young plants, as is shown later, it follows that under such conditions the dangerous season may be greatly prolonged. Where the plants grow on the shores of temporary ponds the growth may not cease until the ponds are entirely dried up. Each bur contains two seeds and it is a well-known fact that ordinarily only one seed germinates in the year following maturity, the other one germinating in the second year or later. So, while the plants are considered as annuals and theoretically can be exterminated if destroyed before the burs are formed, one must expect another crop from the seeds in which germination has been delayed.

EXPERIMENTAL WORK

The experiments on which this report is based were carried on in the years 1920, 1921, 1922, and 1923. Most of the work was done at the Salina Experiment Station, but a few experiments were made at the Bureau of Animal Industry Experiment Station, Bethesda, Md., near Washington, D. C. There were, in all, 67 experiments with swine, 11 with sheep, 12 with cattle, and 19 with chickens. Table 1 contains a summarized statement of these experiments.

TYPICAL CASE OF FIG 18

Pig 18 was a female, 2 months old, of the Poland-China breed. She was in good, healthy condition at the time of the experiment and weighed 40.5 pounds. To this animal was given, at 9.15 a. m., June 9, 1.496 per cent of the animal's weight of *Xanthium echinatum* in the young cotyledon stage. The plants were cut up and mixed with garbage and bran. At 4 p. m. it was noticed a little of the plant remained, but after the addition of more bran the animal ate the remainder.

At the time of feeding, 9 a. m., the pig's temperature was 101.3° F. and respiration 28. At 8 p. m. the temperature was 102, respiration 32. No symptoms appeared during the day. On the morning of June 10 the temperature was 100.6° F., respiration 25, and at 8 p. m. the temperature was 101.3°, respiration 28. During the day the animal was given grass and bran and showed no symptoms of illness. On June 11, at 6.15 a. m., the pig was walking about in her pen, frothing at the mouth, and had been vomiting. Her temperature at 6.18 was 101.4° F., respiration 26. The animal was weak, and at 6.30 she lay down and then got up and walked about the pen in an uneasy manner. At 7.20 she was getting weaker and tottered when attempting to walk. At 8.20 a. m. the temperature was 97° F., respiration 28. At 8.30 the temperature was 97.3°. The animal was lying down with her forefeet drawn up below her jaws. She was gasping for breath and somewhat salivated. The legs were trembling and the respiration spasmodic. At 8.35 a. m. the temperature was 96.1°, respiration 44. The animal continued to gasp, the body moving in a jerky and trembling fashion and occasionally emitted loud grunts. At 8.40 a. m. the temperature was 95.3°. The movements of the legs and the gasps became more continuous. At 9.03 the temperature was 95.7° F., the animal continually gasping, and she died at 9.05 a. m.

Plate III, Figures 1 and 2, show her condition at 8.20 and 8.25 a. m.

In the autopsy the fundus portion of the stomach showed extreme congestion. There was slight congestion in the duodenum and in the cecum. The liver was dark and well filled with blood, but it was not noted, as, in some other cases, that the bile was abnormal. There was some congestion in the abdominal lymph glands.

This animal may be considered typical in the symptoms and rapidity with which the symptoms developed, although the autopsy was not so characteristic as in some other cases.

The lesions, as shown in the microscopic study, were typical in every respect.

DISCUSSION AND GENERAL CONCLUSIONS

SYMPTOMS IN PIGS

The first symptom noted in poisoned pigs, ordinarily, was depression. This was accompanied in most cases with nausea which frequently resulted in continued vomiting. As the sickness progressed the animals became so weak as to be unable to stand. In those that were very sick the respiration became labored, the animals frequently gasping for breath. The pulse became rapid and weak. In some cases there were spasmodic movements of the body. Occasionally the animals moved their legs in what might be described as running movements. Sometimes, however, death resulted without these spasmodic movements. The appearance of sick animals is shown in the pictures of pig 18, Plate III, Figures 1 and 2, and in the series of pictures of pig 21, in Plate IV, Figures 1, 2, and 3.

TABLE 1.—Summary of feeding experiments with *Xanthium echinatum*, 1920 to 1923

Animals		Date of feeding	Method of feeding	Part of plant used	Quantity fed, shown in per cent of animal weight		Place and date of plant collection	Result	Remarks
Designation	Weight				Green	Dry			
Pig 12.....	Pounds 44	June 13-July 9, 1920.	With bran mash.	Whole plant, young cotyledon stage.			Salina, Utah, June 12-July 7, 1920.	Not sick.	On July 2, 3, 7, and 8 no cocklebur was fed.
Pig 10.....	13.50	July 6, 1920.	do.	Dry, mature fruit.			Salina, Utah, June and July, 1920.	do.	Six burs eaten to determine mechanical injury.
Pig 10.....	17.25	July 8, 1920.	do.	Whole plant, young cotyledon stage.	.895		Salina, Utah, July 7, 1920.	Sick.	
Pig 11.....	14.25	July 10, 1920.	do.	do.	.771		do.	Not sick.	
Pig 10.....	17.50	July 13-26, 1920.	do.	do.	7.287		Salina, Utah, July 12-19, 1920.	do.	
Pig 12.....	22	July 21, 1920.	do.	Roots.	2.604		Salina, Utah, July 20, 1920.	do.	
Pig 10.....	20	July 25, 1920.	do.	Seeds from dry, mature burs.		0.158	Salina, Utah, July 27, 1920.	do.	Seeds ground and mixed with bran.
Pig 10.....	21.50	July 29, 1920.	do.	Roots.	1.752		Salina, Utah, July 27, 1920.	do.	Roots finely ground and mixed with bran.
Pig 10.....	23	Aug. 2-3, 1920.	do.	do.	5.046		Salina, Utah, July 31, 1920.	do.	Do.
Pig 11.....	22	Aug. 8, 1920.	do.	Seeds from dry, mature burs.		.685	Salina, Utah, July 31, 1920.	do.	752 seeds.
Pig 10.....	33.75	Aug. 18, 1920.	do.	do.		.275	Salina, Utah, July 31, 1920.	Death.	Burs collected at Salina, Utah.
Pig 12.....	40	Aug. 23, 1920.	do.	Whole plant, young cotyledon stage. ¹	1.102		do.	Not sick.	Do.
Pig 12.....	54.50	Sept. 6, 1920.	do.	do.			Grown at station.	do.	
Pig 12.....	64	Sept. 14, 1920.	do.	Seeds from dry, mature burs.	1.472		Salina, Utah, September, 1920.	do.	
Pig 12.....	74	Sept. 25, 1920.	do.	do.		.2204	Salina, Utah, September, 1920.	do.	
Pig 13.....	15	Feb. 4-5, 1921.	do.	do.		.2335	Salina, Utah, Sept. 19, 1920.	do.	Do.
Pig 14.....	19	Feb. 13, 1921.	do.	Whole plant, young cotyledon stage.	4.850		Grown in greenhouse, Washington, D. C.	Death.	Do.
Pig 15.....	30.50	Mar. 1, 1921.	do.	do.	2.600		do.	do.	Burs collected at Salina, Utah, September, 1920.
Pig 16.....	60	Mar. 15, 1921.	do.	do.	1.763		do.	do.	Do.
Pig 17.....	32.50	Mar. 31, 1921.	do.	Whole plant, medium age, cotyledon stage. ²	1.064		do.	Not sick.	Do.
Pig 17.....	32.50	Apr. 4, 1921.	do.	Whole plant.	1.473		do.	do.	Leaves from 4 inch to 2 inches long. Finely chopped and mixed with bran.
Pig 18.....	40.50	June 9, 1921.	do.	Whole plant, young cotyledon stage.	1.496		Salina, Utah, June 8, 1921.	Death.	500 plants.
Pig 19.....	36.50	June 12, 1921.	do.	Cotyledons, young cotyledon stage.	.676		do.	Not sick.	
Pig 20.....	42.50	June 16, 1921.	do.	do.	.736		Salina, Utah, June 15, 1921.	Symptoms.	682 plants. Animal showed weakness and vomited.



FIG. 1.—PIG 18 AT 8.20 A. M.



FIG. 2.—PIG 18 AT 8.25 A. M.



FIG. 3.—SHEEP 636, SHOWING EXTREME DE-
PRESSION AND WEAKNESS



FIG. 1.—PIG 21 AT 11.44 A. M., SHOWING NAUSEA AND WEAKNESS



FIG. 2.—PIG 21 AT 11.45 A. M., SHOWING WEAKNESS, AS SHOWN BY DIFFICULTY IN HANDLING ITS LEGS



FIG. 3.—PIG 21 AT 12.26 P. M., WHEN COMPLETELY PROSTRATED, JUST BEFORE DEATH

Pig 19	37.75	June 21, 1921	do.	Stems and roots, young cotyledon stage.	1.333	Salina, Utah, June 20, 1921.	Not sick.	882 plants.
Pig 20	50	June 27, 1921	do.	Whole plant, young cotyledon stage.	1.473	Salina, Utah, June 24, 1921.	do.	Plants ground.
Pig 19	44	do.	do.	Cotyledons, young cotyledon stage.	.741	do.	do.	Cotyledons ground.
Pig 20	53.25	July 5, 1921	do.	Whole plant, young cotyledon stage.	1.488	Salina, Utah, July 1, 1921.	do.	
Pig 19	51.5	July 9, 1921	do.	do.	1.496	do.	do.	607 plants.
Pig 20	61	July 11, 1921	do.	do.	1.521	do.	do.	700 plants.
Pig 22	26.25	Aug. 18, 1921	do.	do.	1.473	Grown at station.	do.	Burs collected at Salina, Utah. 235 plants.
Pig 23	24.5	Aug. 24, 1921	do.	do.	1.473	do.	do.	Burs collected at Salina. 243 plants.
Pig 21	16.75	Aug. 29, 1921	do.	Cotyledons, young cotyledon stage.	.772	do.	do.	Burs collected at Salina. 176 plants.
Pig 22	29.25	do.	do.	Whole plant, young cotyledon stage.	1.697	do.	do.	Burs collected at Salina. 301 plants.
Pig 23	29	Sept. 5, 1921	do.	do.	1.540	Salina, Utah, June 16, 1921.	do.	Loss in drying, 92.08 per cent. 337.4 plants.
Pig 21	18.50	Sept. 9, 1921	do.	Cotyledons, young cotyledon stage.	.698	Grown at station.	do.	Burs collected at Salina, 296 plants.
Pig 22	32.50	do.	do.	Whole plant, young cotyledon stage.	2.200	Salina, Utah, June 16, 1921.	do.	Loss in drying, 92.08 per cent. 620.2 plants.
Pig 23	32	Sept. 11, 1921	do.	Cotyledons, medium cotyledon stage.	.882	Grown at station.	do.	473 plants.
Pig 21	21.50	Sept. 14, 1921	do.	Cotyledons, young cotyledon stage.	1.740	Salina, Utah, June 20, 1921.	Death.	Loss in drying, 89.74 per cent.
Pig 23	33	do.	do.	Whole plant, young cotyledon stage.	4.514	Salina, Utah, early in summer.	Not sick.	Loss in drying, 92.15 per cent.
Pig 22	37.50	Sept. 22, 1921	do.	Seeds from green burs.	.220	Salina, Utah.	do.	Bacon grease given; 508 burs. 755 seeds made 37.5 grams.
Pig 23	37.50	do.	do.	Cotyledons, young cotyledon stage.	.882	Grown at station.	do.	908 plants.
Pig 26	25.25	June 29, 1922	With bran and garbage.	do.	.881	Salina, Utah, June 28.	Symptoms.	
Pig 25	29.50	July 5, 1922	do.	Stems and roots, young cotyledon stage.	.881	do.	Not sick.	Cotyledons from plants in 2, 4, and 6-leaf stage.
Pig 24	28.75	do.	do.	Cotyledons	1.329	do.	do.	Do.
Pig 26	27	July 10, 1922	do.	do.	.881	Salina, Utah, July 10.	do.	Do.
Pig 24	31.50	July 14, 1922	do.	do.	.881	do.	do.	Plants, 2, 4, and 6-leaf stage.
Pig 25	32	do.	do.	Whole plant except cotyledons.	.992	Salina, Utah, July 20.	do.	Do.
Pig 25	39.25	Aug. 3, 1922	do.	do.	2.200	Salina, Utah, July 21.	do.	Given 1.5 pints milk after feeding and 1 quart next morning
Pig 24	44.75	Aug. 9, 1922	do.	Cotyledons	1.895	Salina, Utah.	do.	
Pig 28	27	Sept. 12, 1922	do.	Whole plant, young cotyledon stage.	1.653	Sterling, Utah, Aug. 29.	do.	

‡ Leaves less than one-half inch long and more than small buds.

† Leaf buds very small.

TABLE 1.—Summary of feeding experiments with *Xanthium echinatum*, 1920 to 1923—Continued

Animal	Designation	Weight Pounds	Date of feeding	Method of feeding	Part of plant used	Quantity fed, shown in per cent of animal weight		Place and date of plant collection	Result	Remarks
						Green	Dry			
Pig 28		31.50	Sept. 18, 1922	With bran and garbage.	Whole plant, young cotyle- don stage.	1.873		Sterling, Utah, Sept. 14	Not sick	Given 1 quart of milk after feeding and 1 quart next morning. Do.
Pig 29		17	Feb. 27, 1922	With bran and milk.	do	1.873		Grown in greenhouse, Washington, D. C.	do	Given 0.132 pound lard after feeding and same quantity next morning.
Pig 30		16.50	do	do	do	1.873		do	do	Given 60 c c. raw linseed oil after feeding, and same quantity next morning.
Pig 30		22.50	Mar. 28, 1923	With bran mash	do	1.873		do	do	
Pig 31		23	June 25, 1923	With bran flour and garbage.	do	1.102		Salina, Utah, June 23, 1923	do	
Pig 34		20	do	do	do	1.102		do	do	
Pig 31		24	July 3, 1923	do	do	1.102		Salina, Utah, June 25, 1923	do	
Pig 34		21	do	do	do	1.102		do	do	
Pig 31		28	July 19, 1923	do	do	1.322		Sterling, Utah, July 18, 1923	do	
Pig 32		34.50	do	do	do	1.322		do	do	
Pig 32		36.50	July 27, 1923	do	do	1.433		do	do	
Pig 34		25.50	do	do	do	1.433		do	do	
Pig 32		40	Aug. 4, 1923	do	do	1.477		Sterling, Utah, Aug. 2, 1923	do	
Pig 31		33	do	do	do	1.477		do	do	
Pig 32		52.50	Aug. 24-26, 1923	do	do	3.306		Sterling, Utah, Aug. 17, 1923	do	
Pig 32		62	Sept. 5, 6, 8, 10, and 11, 1923	do	do	6.610		Sterling, Utah, Sept. 1, 1923	do	
Sheep 565		80.75	July 8, 1920	Balling gun	Leaves and cotyledons	1.102		Salina, Utah, July 7, 1920	do	4-leaf stage.
Sheep 474		119	July 23, 1920	do	Dry, mature fruit		212	Salina, Utah, July 20, 1920	do	Plant material ground.
Sheep 631		78.50	June 25, 1921	do	Whole plant, young cotyle- don stage.	1.477		Salina, Utah, June 22, 1921	do	
Sheep 636		75.50	June 28, 1921	do	do	1.543		Salina, Utah, June 27, 1921	Death	Plant material ground, 1,043 plants.
Sheep 636		90.75	July 25, 1922	do	Whole plant except cotyle- dons.	1.477		Salina, Utah, July 21, 1922	Not sick	Plants in 2, 4 and 6-leaf stage. Do.
Sheep 699		109	July 29, 1922	do	do	1.543		do	do	
Sheep 711		60	June 21, 1923	do	Whole plant, young cotyle- don stage.	1.102		Salina, Utah, June 20, 1923	do	
Sheep 711		74.25	July 2, 1923	do	do	1.102		Salina, Utah, June 23, 1923	do	

Sheep 711	68	July 19, 1923	do	do	do	1, 322	Sterling, Utah, July 18, 1923.	do	
Sheep 711	70, 50	July 26, 1923	do	do	do	1, 433	do	do	
Sheep 711	72, 50	Aug. 4, 1923	do	do	do	1, 477	Sterling, Utah, Aug. 2, 1923.	do	
Cattle 938	289	June 9, 1922	With green grass.	Whole plants	do	1, 496	Salina, Utah, June 8; grown at station.	do	Most of material young cotyledon stage.
Cattle 954	308	June 16, 1922	With hay	Whole plant, young cotyledon stage.	do	2, 255	Salina, Utah, June; grown at station.	do	
Cattle 938	346	July 31, 1922	do	Cotyledons	do	2, 611	Salina, Utah, July 21	do	From 2, 4, and 6-leaf stage plants.
Cattle 946	500	Aug. 7-11, 1922	do	Whole plant except cotyledons.	do	1, 421	Salina, Utah, Aug. 5	do	
Cattle 954	400	Aug. 21, 1922	With hay and bran.	Whole plant, young cotyledon stage.	do	5, 502	Sterling, Utah, Aug. 16 and 20.	Death	Death in 19½ hours after feeding.
Cattle 945	400	Sept. 8, 1922	do	do	do	2, 911	Sterling, Utah, Sept. 5, 1923.	do	Death in 12 hours.
Cattle 964	230	July 10, 1923	With alfalfa hay	do	do	2, 904	Sterling, Utah, July 4, 1923.	Not sick	
Cattle 964	240	July 19, 1923	do	do	do	2, 425	Sterling, Utah, July 18, 1923.	do	
Cattle 964	250	Aug. 3, 1923	do	do	do	2, 557	Sterling, Utah, Aug. 2, 1923.	do	
Cattle 969	290	Aug. 10, 1923	do	do	do	2, 557	Sterling, Utah, Aug. 17, 1923.	do	
Cattle 997	430	Aug. 22, 1923	do	do	do	2, 700	Sterling, Utah, Sept. 1, 1923.	do	
Cattle 970	555	Sept. 4, 1923	Force fed.	do	do	2, 81	Sterling, Utah, Sept. 1, 1923.	do	
Chicken 37	3, 351	July 20, 1923	do	Mature seed.	do	1, 47	Salina, Utah, June 20, 1923.	Death	Death.
Chicken 39	3, 009	do	do	do	do		do	Not sick	Not sick.
Chicken 35	3, 847	July 23, 1923	do	Whole plant, young cotyledon stage.	do	2	Salina, Utah, July 18, 1923.	do	
Chicken 36	4, 299	do	do	do	do		do	do	
Chicken 34	3, 461	July 26, 1923	do	do	do	3	Salina, Utah, June 20, 1923.	do	
Chicken 33	3, 780	do	do	Mature seed.	do		do	do	
Chicken 38	3, 626	July 30, 1923	do	do	do	18	Salina, Utah, June 20, 1923.	do	
Chicken 35	3, 970	Aug. 4, 1923	do	do	do	20	do	do	
Chicken 36	3, 472	do	do	Whole plant, young cotyledon stage.	do	4	Sterling, Utah, Aug. 2, 1923.	do	
Chicken 33	4, 040	Aug. 7, 1923	do	do	do	5	do	do	
Chicken 34	3, 709	do	do	Mature seed.	do		Salina, Utah, June 20, 1923.	do	
Chicken 37	3, 461	Aug. 10, 1923	do	do	do	22	do	Symptoms	
Chicken 38	3, 897	do	do	Whole plant, young cotyledon stage.	do	6	Sterling, Utah, Aug. 2, 1923.	Not sick	
Chicken 37	3, 406	Sept. 4, 1923	do	Dry, mature fruit	do		Salina, Utah, June 20, 1923.	Slight depression.	6 burs
Chicken 38	4, 078	Sept. 5-6, 1923	do	Whole plant, young cotyledon stage.	do	12	Sterling, Utah, Sept. 1, 1923.	Death	6 per cent daily for 2 days.
Chicken 35	4, 056	Sept. 10, 1923	do	do	do	6	do	Not sick	
Chicken 32	3, 615	Sept. 10-11, 1923	do	do	do	9	do	do	5 grams grease given after feeding.
Chicken 37	3, 174	Sept. 17, 1923	do	Mature seed	do		Salina, Utah, June 20, 1923.	do	12 burs.
Chicken 32	3, 836	do	do	Dry, mature fruit	do		do	Slight depression.	

SYMPTOMS IN SHEEP AND CATTLE

In sheep and cattle, as in the pigs, the first effect of the plant was depression. This was followed by extreme weakness with labored respiration, gasping, and spasmodic movements of the limbs. Plate III, Figure 3, shows this condition of extreme depression in sheep 636. In some cases there was evidence of hyperesthesia and in the cattle there was a peculiar trembling or quivering of the muscles. Vomiting was not noted in either cattle or sheep.

SYMPTOMS IN CHICKENS

The symptoms in chickens were not especially characteristic. As in the other animals there was marked depression, followed by weakness and in one case, chicken 33, a distinct condition of coma. There were no spasmodic movements, although in one case there was some struggling just before death.

AUTOPSY FINDINGS

Autopsies were made on 7 pigs, 1 sheep, 2 cattle, and 2 chickens.

The pictures presented by these examinations were not of a uniform character in details but showed some outstanding features. The parts especially affected were the alimentary canal, the liver, and to a lesser degree, other glandular structures.

In the pigs the walls of the stomach were more or less congested, the stomach congestion in the cattle being confined to the fourth stomach. This congestion extended, in some cases, to a greater or less degree, to the duodenum, jejunum, ileum, cecum, and colon. Where this congestion occurred there was a tendency to thickening of the walls with a serous infiltration.

In the sheep and pigs the liver was congested or spotted, while in the cattle it was bluish. The walls of the gall bladder, and in some cases those of the common bile duct, were thickened and the bile was thick and viscid.

The spleen in some cases appeared congested and a similar condition was found in some of the lymph glands, especially those of the abdominal cavity.

In some cases there was an accumulation of a serous infiltration about the gall bladder and ducts and there was generally an excess of serum in the abdominal cavity. These conditions varied in the different animals and did not appear uniformly by any means; this was especially true of the pigs the only abnormal condition common to a majority being the congested stomach.

The cattle presented a more nearly uniform picture, each animal having a congested fourth stomach, bluish liver, thickened gall bladder, congested spleen, serous infiltration about gall bladder and ducts, and abnormal suprarenals. It should be noted, however, that the thickened gall bladder may have been due, in part at least, to the flukes with which the cattle were infested.

In one of the two chickens autopsied there were no abnormal conditions; in the other there were petachiae on the heart, an unusual quantity of pericardial fluid, the serous fluid of the abdominal cavity was bloody, the crop showed slight inflammation, and the gall bladder was distended.

MICROSCOPIC CHANGES IN TISSUES

At the time of the autopsies, samples were preserved of such tissues as it was thought desirable to examine in greater detail. In this work 142 samples of tissues were sectioned and studied.

In the sections of the livers every lobule examined was found to be hemorrhagic, although the interlobular branches of the portal vein did not contain an excess of blood. Usually most of the intra-lobular capillaries and often the central lobular veins had more or less completely disappeared, and many of the hepatic cells were necrotic. In the most advanced cases the liver cords were broken up, leaving a few dead hepatic cells lying in a mass of blood. In the least advanced cases the hepatic cells were still arranged in cords, but were compressed, and most of them necrotic. Usually two or three rows of cells in the peripheral portion of the lobule were less severely affected, though greatly swollen. In 6 of the 10 cases an excess of leucocytes was found throughout the liver tissues.

The bile ducts varied considerably in their condition. As a rule the smallest ducts were little altered. The larger ducts usually had a swollen epithelium, a few cells of which were necrotic. A few ducts, in four of the cases, contained exfoliated epithelial cells. In six of the cases the interlobular connective tissue was edematous, and in two cases the capillaries of this region were congested. In four cases the walls of the gall bladders were necrotic and thickened by a serous exudate. In three of these the tissues were infiltrated with leucocytes, and in one case there were hemorrhages.

The changes in the kidneys were less severe than those in the livers. The most pronounced alterations were in the epithelium of the convoluted tubules. This was swollen and often in a well-advanced stage of granular disintegration, the granular material being scattered throughout the lumina. In such tubules the nuclei were disarranged and often shrunken. In some cases many cells were necrotic. Similar changes had occurred in the ascending limb of Henle. The descending limb was often distended and sometimes contained hyaline casts. The collecting tubules were much less affected, but in some cases they had a swollen and even granular epithelium. Blood, as a rule, was not abundant, though occasional capillaries, both inter-tubular and those in the glomeruli, were distended, and in some cases minute hemorrhages were present.

The sections of the walls of the fundus portion of the stomach of the pigs, with the single exception of pig 13, showed a capillary congestion or hemorrhagic condition of the mucous membrane. In the milder cases this consisted of a pronounced distention and engorgement of the capillaries near the surface of this membrane. In the more pronounced cases severe hemorrhages had occurred. The congestion and hemorrhage were confined, in all but one case, to the portion of the mucous membrane next to the stomach cavity, and extended through about one-third of its thickness. Usually there was edema of the interglandular tissues, with a swelling or degeneration of the cells.

No congestion or hemorrhage was found in the mucous membrane of the sections of the abomasum of the sheep or cattle. In one of the cattle the interglandular cells were swollen, and in this and the other cases leucocytes in this region were slightly more abundant

than normal. Other layers of the walls of the stomachs of all animals appeared to be unaffected.

No uniform pathological change was noted in any portion of the intestines. In very few instances did the sections show a congested condition. In most sections from the various portions there was an excess of lymphoid cells or other leucocytes in the mucous membrane. In some instances, however, this was probably due to parasites. In half of the samples examined the serosa was thickened or contained an excess of leucocytes. Some of them were of the endothelial type and were on the surface of the serosa, probably due to the presence of an irritant in the abdominal cavity.

While there was evidence of some irritation in the spleen it was not found uniformly in all the animals, nor was it particularly severe. Of the 10 cases studied, 5—2 pigs, the sheep, and both cattle—had congested spleens. This blood, however, was very unevenly distributed, some small areas being full of red blood corpuscles, while in other areas there were few, if any. In 5 cases there was a swelling of the pulp reticular cells, and in 6 cases splenic cells seemed unusually abundant. In the sheep and in both cattle the lymphoid cells of the germinal centers had undergone more or less degeneration. In the most extreme case—cattle 954—the nuclei of these cells were found in various stages of degeneration. Many of them were fragmented, and some of the fragments had been taken up by phagocytes.

Sections of several mesenteric and mediastinal lymph glands were examined. These were in a condition very similar to that found in the spleens. In 8 glands taken from 5 of the animals the reticular cells were distinctly swollen and in 5 of these glands they were noticeably degenerated. In 5 glands there was an excess of large mononuclear cells of the endothelial cell type. Only rarely were the glands congested. In 2 cases small hemorrhages had occurred in the lymphoid areas and in 3 cases red blood corpuscles were found in the lymph spaces. In 1 pig and the sheep polymorphonuclear leucocytes were present in considerable excess. In the glands of both cattle eosinophiles were present in excess, a condition doubtless connected with the fluke infestation of the livers of the animals.

Little of significance was found in sections of the lungs or of heart tissue. On the average there was a relatively small quantity of blood in the interalveolar capillaries, many of them being entirely empty. The sheep was the only animal in which a congestion was found. Occasionally the epithelium of the bronchial tubes was swollen and sometimes the surrounding connective tissue was somewhat congested. In the walls of the ventricles occasional areas of capillary congestion were found, but the condition was not constant.

In general, then, it appears that the most severe and perhaps the primary effect was in the liver, and had resulted in hemorrhages in all the lobules and a severe necrosis of the hepatic cells. The epithelium of the larger bile ducts was often injured, the change extending apparently to the gall bladder, the walls of which were sometimes necrotic. In the kidneys there was a condition of parenchymatous nephritis affecting mainly the convoluted tubules and the ascending limb of Henle with less injury to other tubules. The mucous membrane of the stomach, in the pigs especially, was congested or even hemorrhagic.

Of the other organs the spleen and lymph glands appear to have shown the effects of a mild irritant. The lungs were characterized rather by a lack of blood than by congestion. This may have been due largely to the removal, from the general circulation, of the considerable quantity of blood which was accumulated in the liver.

TOXIC AND LETHAL DOSAGE

As is shown elsewhere, the plant was found to be poisonous only in the younger stages; the largest part of the experimental work was done, therefore, on that stage of the plant which has been designated as the young cotyledon stage. This means the stage of the plant up to the time the first pair of leaves is partially formed. In gathering the plant for experimental purposes, especially during the later feedings, great care was taken to get it as young as possible. Plants in which the first pair of leaves was much developed were not ordinarily used. In the following discussion of dosage, only plants of this age are considered.

In the experiments on pigs the smallest toxic dose was 0.736 per cent of the weight of animal in pig 20; pig 10 was made sick on 0.895 per cent. The smallest lethal dose was 1.496 per cent of animal, in pig 18. When no remedy was given and the plant was fresh, the largest dose given without effect was in the case of pig 20, July 11, 1921, being 1.521 per cent. (Pig 12 received June 13, without effect, 2.856 per cent of its weight. The plant in this case, however, was probably older than the young cotyledon stage.)

In general, then, it may be stated that the lethal dose for pigs is about $1\frac{1}{2}$ per cent of the weight of the animal, and that the toxic dose may be as small as half of that quantity.

Sheep 636 was killed by 1.543 pounds per hundredweight of animal, while sheep 631 and sheep 711 ate 1.477 pounds with no harm. So far as these experiments go, it would appear that the plant is about equally toxic to pigs and sheep.

Cattle 945 was killed by 2.911 pounds per hundredweight of animal, while cattle 970 received 2.801 pounds per hundredweight of animal with no effect. It seems probable from these results that the toxic and lethal dose for cattle is about 3 pounds per hundredweight of animal, and that it takes about twice as much to poison cattle as it does to affect pigs or sheep.

With the chickens the feedings gave no definite results as to the toxic and lethal doses. Chicken 38 on two dates, a month apart, received 6 per cent of its weight and was not affected. Chicken 33 was fatally poisoned by two doses of 6 per cent on two successive days.

In the autopsy of chicken 33 the crop was found still distended by a mass of *Xanthium* which was thought to be fully equal to the quantity given on the second day of the feeding. While the data are insufficient for a definite statement, these experiments indicate that the toxic and lethal dose is between 6 and 12 per cent of the animal's weight, with the probability that it is much nearer 6 than 12. It is evident that chickens are much less susceptible to *Xanthium* poisoning than are pigs, cattle, or sheep.

The relative toxicity of different parts of the plant is discussed on page 20.

TIME FROM FEEDING TO APPEARANCE OF SYMPTOMS

TABLE 2.—Time elapsed from feeding to development of symptoms

Animal	Date of feeding	Time before symptoms
		<i>Hrs. Ms.</i>
Pig 10.....	Aug. 18, 1920.....	21 45
Pig 15.....	Mar. 1, 1921.....	24 00
Pig 18.....	June 9, 1921.....	38 15
Pig 20.....	June 20, 1921.....	43 10
Pig 21.....	Sept. 14, 1921.....	19 15
Sheep 636.....	June 28, 1921.....	16 35
Pig 26.....	June 29, 1922.....	17 35
Cattle 944.....	Aug. 21, 1922.....	14 55
Cattle 945.....	Sept. 8, 1922.....	8 35
Chicken 39.....	July 20, 1923.....	17 15
Chicken 37.....	Aug. 10, 1923.....	16 50

Table 2 is far from giving the exact time which elapsed between the feeding and the development of symptoms. In some cases the feeding continued over a considerable period, so that there may have been some poisonous effects even before the conclusion of the feeding. The time is computed from the end of the feeding. There is some indefiniteness in the time at which symptoms developed. The animals were not, in all cases, under constant observation, and in some the first symptoms were noted when the animals were observed in the morning, and it is possible that they had been sick for a considerable period before that time.

The shortest time noted was in the case of cattle 945—8 hours and 35 minutes. The longest period was in the case of pig 20—43 hours and 10 minutes. In most cases, however, the time was a little less than 24 hours.

In general, a period of several hours elapses before symptoms appear, and this time, in some cases, may be between one and two days.

It should be noted that the chickens quoted in Table 2 received seeds, while the other animals, with the exception of pig 10, August 18, received plants in the young cotyledon stage. This difference in material apparently did not affect the time elapsing between the feeding and the development of symptoms.

DURATION OF SICKNESS

Table 3 shows the duration of illness in cases of animals that either were sick or died.

TABLE 3.—Duration of sickness

Animal	Date of feeding	Time sick	Result
		<i>H. M.</i>	
Pig 10.....	July 8, 1920.....	6 00	Recovery.
Pig 10.....	Aug. 18, 1920.....	2 10	Death
Pig 15.....	Mar. 1, 1921.....	2 27	Do.
Pig 18.....	June 9, 1921.....	2 50	Do.
Pig 20.....	June 16, 1921.....	23 40	Recovery.
Pig 21.....	Sept. 14, 1921.....	2 25	Death.
Pig 26.....	June 29, 1922.....	1 35	Recovery.
Sheep 636.....	June 28, 1921.....	13 00	Death.
Cattle 944.....	Aug. 21, 1922.....	3 19	Do.
Cattle 945.....	Sept. 8, 1922.....	0 40	Do.
Chicken 39.....	July 20, 1923.....	7 23	Do.
Chicken 37.....	Aug. 10, 1923.....	24 00	Recovery.
Chicken 33.....	Sept. 6, 1923.....	13 10	Death.

In Table 3 the time was computed from the first to the last-noted symptom. It is evident that in many cases this does not show the actual duration of illness. Inasmuch as the animals were not under constant observation, they were in some cases undoubtedly sick before the first symptom was noted, and doubtless in many cases the illness continued after the last-noted symptom. In some the first symptom was noted in the morning after a night during which no observations were taken. This accounts for the very short time of cattle 945 because when the first symptom was noted in the morning the animal was already very sick. Table 3, however, gives in a general way a fair indication of the time during which the animals were sick. Excluding cattle 945, the shortest period of illness was in the case of pig 26 which was sick 1 hour and 35 minutes and recovered. The longest period which any animal was sick was in the case of chicken 37, which recovered after an illness of 24 hours.

If the time of symptoms recorded is compared with the dosage which the animals received it will be seen that there is no clear correlation between them.

EFFECT OF CONTINUED FEEDING

Three pigs were fed *Xanthium* daily for a considerable period. Pig 12 was fed from June 13 to July 9, with the exception of four days, receiving daily doses varying from 0.313 to 2.856 per cent of its weight. On June 23 it received 2.125 per cent, June 24, 1.966, June 25, 1.574, and June 26, 1.574. On June 28 and June 29, and on July 4, 5, and 6, it received daily 1.574 per cent of its weight.

Pig 10, from July 13 to 20, with the exception of one day, received the plant daily in doses varying from 0.378 per cent of its weight to 1.749 per cent. On July 17 it received 1.512 per cent of its weight and on July 18, 1.749 per cent, with no effect.

Pig 32 from August 25 to August 26 received daily doses of 1.102 per cent of its weight, and on September 5, 6, 8, 10, and 11 received 1.333 per cent.

It has been found, as shown on page 15, that the minimum toxic dose of the plant, in the young cotyledon stage, is 0.736 per cent of the pig's weight, and the minimum lethal dose is 1.496 per cent. These animals then, in some cases, received on successive days not only more than the toxic dose but more than the lethal dose. With the exception of pig 12, which received on the first day 2.856 per cent of its weight, all the animals received in the first feedings less than the toxic dose. All these feedings were of the plant in the young cotyledon stage. After it was found that the plant was most poisonous before the growth of the leaves, great care was used to collect for the routine feeding only the very young plants. Less attention was paid to this in the early part of the work; while the notes are incomplete in regard to the collections used, it is known that some of the material used for pigs 10 and 12 was in the 2 and 4 leaf stages. This probably explains the failure to poison pig 12 on 2.856 per cent of its weight. Making some allowance for the indefiniteness in regard to the age of the plant in some of the feedings, it is still noticeable that pigs 10 and 12 on successive days could receive such large quantities with no resulting symptoms.

In the case of pig 32 it is known that all the feedings were of the young plant, before the development of the leaves, and this animal repeatedly received a quantity slightly less than the minimum lethal dose, with no ill effect. Twice on two successive days it received a total of 2.645 per cent of its weight, while, with the exception of pig 12 on June 9, as has been explained, no other pig received more than 1.521 per cent without toxic effects. These cases make it fairly evident that *Xanthium* is not a cumulative poison, but that, on the other hand, it is rather rapidly eliminated.

Inasmuch as several times, after a series of daily feedings, a single feeding much greater than the minimum lethal dose produced no symptoms, it seems probable that some toleration was acquired. The experiments were not sufficiently numerous so that a positive statement can be made of this fact, but it seems probable that this is the case.

ANIMALS POISONED BY COCKLEBUR

The experimental work described in this bulletin was on swine, sheep, cattle, and chickens and demonstrated conclusively the poisonous effect of cocklebur on these animals. There was no work done by the writers on horses, but others have reported such cases of poisoning. It is presumable that they would be affected if they ate the plant. As a matter of fact, horses are less liable than other domestic animals to be injured by any poisonous plants, because they select their feed with more care. The dosage would indicate that while chickens may be poisoned, under ordinary circumstances such occurrences are rare.

PART OF PLANT POISONOUS

Experimental feedings were made not only of the whole plant in the young cotyledon stage but of seeds, roots, cotyledons, stems and roots, and plants with the cotyledons removed. It was found that plants above the young cotyledon stage were only slightly poisonous and these are therefore of no practical importance from the standpoint of the toxicology of the plant. Moreover, as the plants in the young cotyledon stage have no disagreeable taste, they are eaten quite readily. As the plants grow older the leaves develop a very bitter taste, and animals generally refuse to eat them.

It was found that the seeds, removed from the burs and fed to some of the pigs, were much more toxic than other parts of the plant. Pig 10 was killed by 0.275 per cent of animal weight. Pig 12 received 0.2535 per cent of animal weight with no effect. It seems probable from the experiments with pig 10 that the minimum toxic and lethal dose is not far from 0.275 per cent of animal weight, although an animal may receive as much as 0.2535 per cent with no apparent effect.

Chicken 37 was made sick on 0.22 per cent of its weight, and chicken 39 died from the same dosage. So far as these few experiments go the seeds appear to be more toxic to chickens than to pigs.

The feeding of plants having 2, 4, and 6 leaves was not attended with harmful result, showing that the plant loses its toxic properties very rapidly as it develops from the cotyledon stage. So far as the experiments indicate it would appear that the cotyledons have about the same toxicity as the whole plant in the young cotyledon stage.

ARE THE POISONOUS PROPERTIES OF THE COTYLEDONS DERIVED FROM THE SEEDS?

Since the seeds are by weight much more poisonous than the young plants, and as the plants, early in their growth, lose their toxic properties, the question has arisen whether the young plants derive their toxicity solely or largely from the seeds. If the plants contain simply the toxic material from the seeds, animals should be poisoned by a number of plants equal to the number of seeds which produce a poisonous effect. Counts were made in 5 experimental feedings to pigs of seeds, in 11 feedings of the whole plant in young cotyledon stage, and in 6 feedings of cotyledons.

The results were as shown in Table 4:

TABLE 4.—*Number of seeds, plants, and cotyledons producing toxic and nontoxic effects to pigs*

Part of plant			Number of seeds per hundred-weight of animal
Seeds.....	Minimum.....	Fatal.....	2,200
	Maximum.....	Nontoxic.....	1,800
Whole plant.....	Minimum.....	Fatal.....	900
	Maximum.....	Nontoxic.....	1,200
Pairs of cotyledons.....	Minimum.....	Toxic.....	1,600
	Maximum.....	Nontoxic.....	1,600

Since in the case of the seeds no effect was produced by 1,800, it may be assumed that 2,200 is not only the lethal dose but is not far from the toxic dose.

Of the whole plant, as there were 11 feedings, 9 of which were between 900 and 1,200, it seems probable that 900 is very close to the minimum toxic and lethal dose.

With the pairs of cotyledons, 1,600 produced symptoms and in another case the same number had no effect, so here, too, the dosage must be close to the minimum. While too much reliance must not be placed on the comparatively small number of experiments, nevertheless the results have some significance, but do not show the expected correlation. On the contrary, considering numbers only, the whole plant is most toxic, the cotyledons next, and the seeds least of all, the proportionate toxic doses being 9, 16, and 22. As the writers have no knowledge of the chemistry of the plant, it is impossible to explain this fact. It is possible, of course, that the toxic substance is not actually greater in the plant, but is in a more available form, but it is also possible that a greater quantity is secreted in the plant. These figures also show quite clearly that the cotyledons do not contain all the toxic substance, as it takes 1,600 cotyledons to equal the effect of 900 whole plants.

MECHANICAL INJURY BY BURS

As stated in the historical résumé, some writers claim that the burs injure animals either by irritation caused by the spines, or by obstruction of parts of the alimentary canal, or by both. There was little evidence in the experiments made by the writers either in sup-

port or in refutation of this claim. In one case, pig 10, July 6, 1920, six fruits of the *Xanthium* were eaten with no deleterious results. Presumably they were digested.

The experimental feeding of fruit to chickens may have some bearing on this question. Six mature burs coated with a stiff mixture of white flour and water were given to chicken 37. The dough was used to cover the spines in order to protect the chicken's throat. During the first 18 hours after feeding, 4 of the burs had disappeared from the crop. Of the two remaining burs one had gone in 90 hours, while the other did not move on for 186 hours.

The first morning following the feeding the chicken was slightly depressed, but by evening the depression was almost entirely gone. At the end of the first 96 hours the animal's weight had dropped 250 grams. After this time it rose slightly, but still remained about 200 grams below the weight at the beginning of the feeding. After the last bur had gone the weight increased. The autopsy showed no effect of the burs.

Chicken 32 was given 12 mature burs which were likewise coated with dough. Eighteen hours after feeding there still remained a mat of burs in the crop, possibly 8 or 9 in number; the clump was too large for counting. Forty-two hours after feeding, the clump of burs was still present, but a few were separated from the main ball. Sixty-six hours after feeding, 6 burs could be counted. These were separated from one another. Ninety hours after feeding, 4 burs still remained. In 97 hours only 1 bur remained and that one had disappeared in 114 hours. At the end of 42 hours the animal was slightly stupid, and the points of his comb were dark. There was a drop in weight of about 70 grams for a week following the feeding.

No serious consequences resulted from these two feedings, but in each case it seems likely that there was a slight disturbance of a general nature. As soon as the cause of the trouble was removed there seemed to be prompt improvement.

While the feces of these animals were not collected, they were under constant observation and it is believed that the burs were digested.

To sum up the present state of our knowledge in regard to injuries to animals from the use of the whole fruit, the statements made by other authors, quoted on pages 2-5, seem to be based on actual experience, and must be taken at their face value. It must be presumed, then, that swine may be injured by the fruit. The experience of the authors, however, indicates that swine do not readily eat the burs and that injuries from this source, if they occur, are somewhat rare. So far as chickens are concerned it is improbable that, when left to themselves, they would ever take as many burs as were given in the experiments, and as these animals were not injured it may be fairly concluded that burs, under ordinary circumstances, do not harm chickens.

TOXICITY OF DRIED PLANT

There were four experimental feedings of dried plant. In these feedings, estimating the material as green plant, pig 21 was killed with 1.74 per cent of its weight of cotyledons. Pig 23 received of the plant in the young cotyledon stage at one time 1.54 per cent of its

weight, and at another 4.514 per cent, and pig 22 received of the plant in the same stage 2.2 per cent; these feedings to pigs 23 and 22 produced no toxic effect. As 1.512 per cent was the largest quantity of the fresh plant which failed to produce symptoms, it seems probable that the plants lose some of their toxicity in drying.

REMEDIES

To avoid losses from *Xanthium*, the most important thing is to prevent animals from eating the weed. Very hungry animals will eat almost anything available. If there is a shortage of good forage, and animals find the young plants of cocklebur, they may easily eat enough to cause serious results.

Pigs are attracted to the margins of shallow ponds, where cockleburs grow in profusion, and are especially liable to be poisoned, and other animals may eat the succulent young plants when there is a lack of other forage. These young plants, as stated, do not have the bitter taste which is found in later growth, and it is not strange that they are eaten in considerable quantity.

In regard to medicinal remedies, considerable work has been done, and the results apparently indicate methods of treatment which may be used successfully. Mrs. A. Creecy, of Raton, N. Mex., told the senior writer that her people had found that if pigs were given whole milk they were not poisoned by cocklebur. Acting on this suggestion, several experiments were made. Pig 28, on September 12, was given 1.353 per cent of animal weight of the young cotyledon stage of cocklebur. It received $1\frac{1}{2}$ quarts of fresh milk, part immediately after the feeding of cockleburs and part the next morning, and showed no symptoms of poisoning. On September 18 the same pig received 1.873 per cent of animal weight of cocklebur. Immediately after the feeding it was given 1 quart of fresh milk and another quart the next morning. Again it showed no symptoms. This experiment was repeated with pig 29, with the same result.

Remembering that 1.5 per cent of animal weight may be considered as the toxic or lethal dose, and that, as stated, the largest quantity of the plant in the young cotyledon stage which was eaten with no toxic results, when no remedy was given, was 1.521 per cent of animal weight, it would seem that the milk must have had a beneficial effect.

Assuming that the beneficial effect of the milk was due to the contained fats, it was thought that similar results might be obtained by the use of other fats or oils. Pig 30, February 27, received 1.873 per cent of animal weight of *Xanthium*, this as before being considered a toxic dose, and immediately after the feeding was given 60 grams (2.1 ounces) of lard and the same quantity the next morning. That quantity of lard was used, although it is about double the quantity of the fat in a quart of rich milk. The animal showed no symptoms of poisoning.

The same animal in another experiment, March 28, received the same quantity of *Xanthium*, and after feeding was given 60 cubic centimeters (2 fluid ounces) of raw linseed oil and the same quantity next morning. As in the use of lard, the dosage of oil was estimated as being about twice the quantity of fat in a quart of milk. In this case, too, no toxic effect followed.

Chicken 37 was given 0.3 per cent of its weight of mature seeds and immediately after the feeding received 5 grams (0.2 of an ounce) of bacon grease: no symptoms followed. As 0.22 per cent produced symptoms in a preceding feeding, and the same quantity in the case of chicken 39 resulted in death, it is probable that the grease was instrumental in preventing poisoning.

The uniformly successful results of these experiments make it probable that in fats and oils, like milk, bacon grease, lard, and linseed oil, we have a distinctly valuable remedy.

SUMMARY

While there have been many reports of the poisoning of animals—more especially of pigs—by the cocklebur, there has been little experimental proof of the toxic character of these plants.

Experimental feedings have shown conclusively that cocklebur plants are poisonous to swine, cattle, sheep, and chickens.

While the burs may produce some mechanical injury and the seeds are very poisonous, stock poisoning is caused by feeding on the young plants before the development of the leaves.

The toxic dosage has been worked out together with the symptoms and lesions produced by the plant.

It has been shown that beneficial remedial effects may be produced by the use of milk, oils, or fats.

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